

P wave of electrocardiogram in early ischaemic heart disease

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The P wave 'terminal force' in lead V₁ of the electrocardiogram has been measured in 159 patients with early ischaemic heart disease and in 206 normal subjects. For subjects over the age of 40, a P wave terminal force more negative than -0.020 mm sec was found in under 1 per cent of the normals and in 18 per cent of the patients with ischaemic heart disease. The P wave terminal force was more negative than -0.030 mm sec in none of the normals and in 7 per cent of the patients. In the group with ischaemic heart disease the QRST patterns were normal in 17 of the 27 with a P wave terminal force more negative than -0.020 mm/sec and in 5 of the 11 with terminal force more negative than -0.030 mm sec. It is concluded that the P wave may be the first component of the electrocardiogram to become abnormal in ischaemic heart disease. A P wave terminal force more negative than -0.020 mm sec in an apparently normal subject should lead to the suspicion of this diagnosis and further investigation.

P wave abnormalities of the electrocardiogram after myocardial infarction are well recognized (Master, 1933; Bloom and Gilbert, 1942; Sutnick and Soloff, 1962; Sitar, 1965; Heikkilä, 1967; Grossman and Delman, 1969; Heikkilä and Luomanmäki, 1970), but little has been written about P waves in ischaemic heart disease before overt infarction has occurred. Our attention was drawn to this subject by the observation that progressive 'mitralization' of the P waves sometimes was seen as the only abnormality of the electrocardiogram in the preinfarction phase of ischaemic heart disease, even a year before infarction. The possibility of using the P wave abnormality as an early warning sign of ischaemic heart disease was supported by finding it in some cases of angina pectoris where the electrocardiogram was normal in other respects.

In order to assess the clinical value of the sign, the P waves in a group of patients with early ischaemic heart disease and in a group of normal controls were examined. Since paroxysmal supraventricular tachycardia may be associated with ischaemic heart disease and/or P wave abnormalities (Davies and Ross, 1963), we have also studied a group of patients with paroxysmal supraventricular tachycardia who were free from evidence of ischaemic heart disease.

Subjects and methods

A total of 159 patients with early ischaemic heart disease was studied. The clinical findings in this group have been described elsewhere (Nixon and Bethell, 1972). No cases with hypertension, previous myocardial infarction, valvular heart disease, or cardiac failure were included. The diagnosis of ischaemic heart disease was based on the history of angina pectoris in 111 patients, in whom the resting electrocardiogram was abnormal in 21 (18%). In the remaining 48 patients without angina pectoris, the diagnosis was made on the basis of electrocardiographic abnormalities at rest (24 cases), after exercise (17 cases), or in the year before or after the first attendance at the cardiac department (7 cases).

The series of normal subjects consisted of 206 people seen in the cardiac department and considered to have normal hearts.

The paroxysmal supraventricular tachycardia series consisted of 43 patients with this clinical diagnosis, none of whom had evidence of ischaemic heart disease, hypertension, valvular heart disease, or Wolff-Parkinson-White syndrome.

In each case, a full history was taken, physical examination performed, and a 12-lead electrocardiogram recorded using a Cambridge direct writing electrocardiograph. Abnormalities of the QRST complexes were noted on the basis of the Minnesota Code (Blackburn *et al.*, 1960). The P wave in lead V₁ was examined with a lens and graticule, measurements being made to the nearest millimetre in amplitude and the nearest 0.01 sec in duration. The size of the 'terminal force' of the P wave in lead V₁ was calculated by

the method of Morris *et al.* (1964). The measurement and calculation of this terminal force are illustrated in Fig. 1. The terminal force is the product of the amplitude in mm and the duration in seconds of the terminal portion of the P wave. Upward deflections are denoted positive and downward deflections negative, e.g. in Fig. 1, the force is $a \times t$ mm sec. In patients with left heart disease involving the left atrium, such as mitral stenosis, progression of the disease results usually in increasing negativity of the P wave terminal force, referred to here as 'mitralization'. Recovery from an illness such as myocardial infarction may be associated with decreasing negativity of the terminal force (Fig. 2).

The majority of patients with ischaemic heart disease had simultaneous apex cardiograms and phonocardiograms performed by a technique described elsewhere (Nixon, Hepburn, and Ikram, 1964), in order to detect and record the atrial gallop, which may be the earliest detectable physical sign of left ventricular dysfunction from ischaemic heart disease.

Results

The terminal portion of the P wave in lead V1 has been recorded as positive, isoelectric, or negative. The incidence of the different P wave morphologies for each diagnostic group in each age range is shown in Tables 1 to 3. A negative 'terminal force', that is a biphasic PV1, was found in about two-thirds of normal subjects over the age of 40 and in the ischaemic heart disease group.

When the magnitude of this terminal force is examined, differences emerge between the two groups. In each age range, the average P wave terminal force in ischaemic heart disease is much more negative than in the normal and paroxysmal supraventricular tachycardia groups, and the negativity increases steadily with age (Fig. 3).

The terminal force for every subject in the study is illustrated in Fig. 4. None of the paroxysmal supraventricular tachycardia group and only one of the normal subjects had a force more negative than -0.020 mm sec. For those over the age of 40, this represents an incidence of less than 1 per cent for the normal subjects (1 out of 113). Of the 152 ischaemic heart disease patients over the age of 40, the P wave terminal force was more negative than -0.020 mm sec in 27 (18%) and more negative than -0.030 mm sec in 11 (7%). In 17 of the 27 patients with a force more negative than -0.020 mm sec and in 5 of the 11 with a force more negative than -0.030 mm sec, there was no other abnormality of the resting electrocardiogram.

Though the P waves were normal in the paroxysmal supraventricular tachycardia

Lead V1

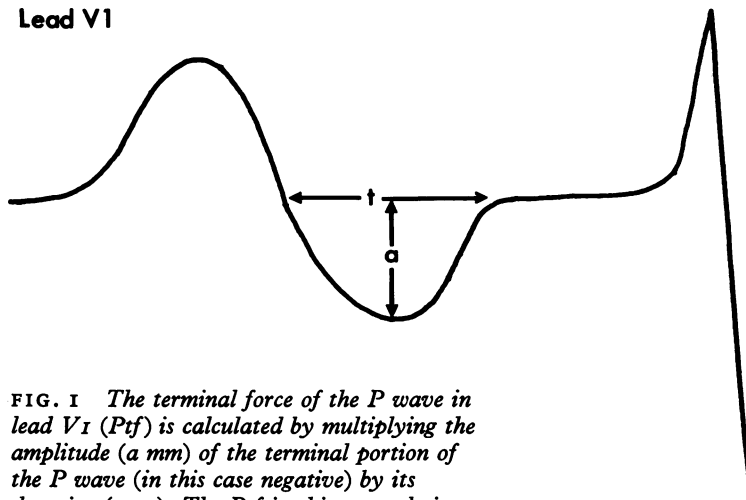


FIG. 1 The terminal force of the P wave in lead V1 (Ptf) is calculated by multiplying the amplitude (a mm) of the terminal portion of the P wave (in this case negative) by its duration (t sec). The Ptf in this example is $a \times t$ mm sec.

FIG. 2 Lead V1 from the serial electrocardiograms of a patient recovering from myocardial infarction complicated by pulmonary oedema. There is gradually decreasing negativity of the P wave terminal force (Ptf) from -0.160 mm sec on 27 May 1970 to -0.018 mm sec on 9 July 1970.

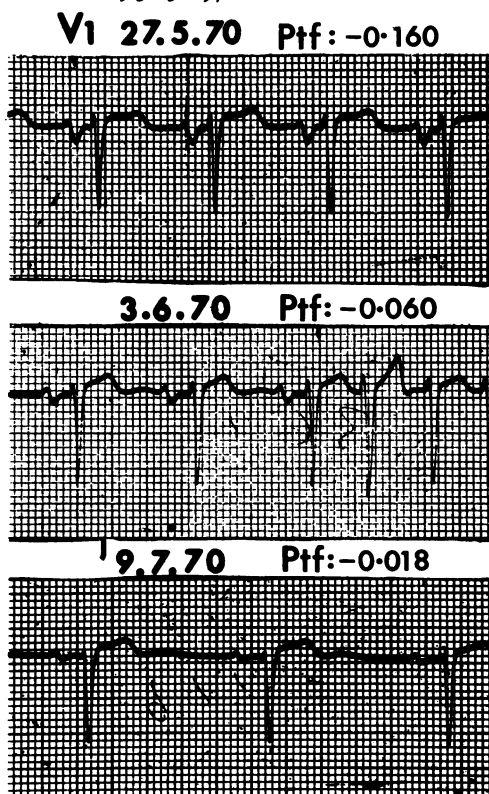


TABLE 1 *P wave morphologies in normal subjects* (see text)

Age range	18-29	30-39	40-49	50-59	60+	Total
No.	50	43	49	41	23	206
% -ve Ptf	42	51	67	61	66	56
% iso. Ptf	42	37	31	29	30	34
% +ve Ptf	16	12	2	10	4	10

Ptf, P wave terminal force.

group, abnormalities were found when tachycardia was combined with ischaemic heart disease. Of the 159 ischaemic heart disease group, 8 gave a history of paroxysmal tachycardia and 4 (50%) of these had a P wave terminal force more negative than -0.020 mm sec and 2 (25%) had a P wave terminal force more negative than -0.030 mm sec. The average terminal force of the patients with both paroxysmal supraventricular tachycardia and ischaemic heart disease was -0.020 mm sec as opposed to -0.010 mm sec for the whole ischaemic heart disease group.

Simultaneous apex cardiography and phonocardiography were performed in 108 of the ischaemic heart disease group. An atrial gallop was detected by these techniques in 71 cases (66%). The incidence of an atrial gallop in patients with a P wave terminal force more negative than -0.030 mm sec was not different from that of the whole ischaemic heart disease group.

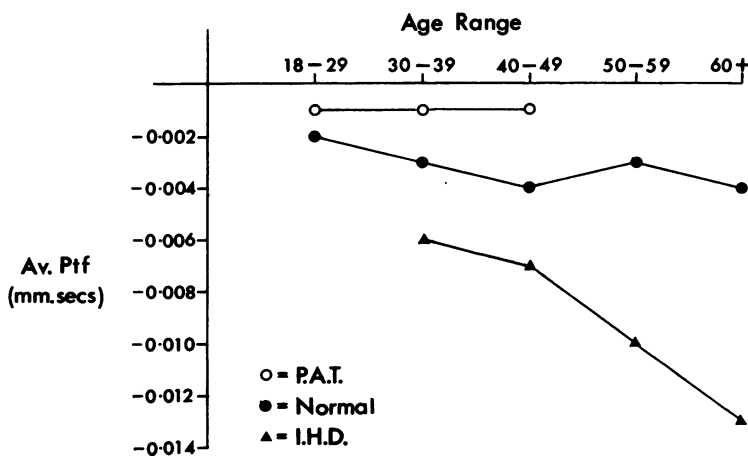
Discussion

Sodi-Pollares and Calder (1956) said that the P wave might be the first component of the electrocardiogram to become abnormal in hypertension, and his observation has been confirmed (Tarazi *et al.*, 1966). Our results suggest that P wave abnormality may also be the first electrocardiographic sign of ischaemic heart disease. P wave changes after myocardial infarction, with increased negativity of the P wave terminal force, are well recognized and have usually been thought to indicate severe heart disease with left ventricular failure (Sitar, 1965; Grossman and Delman, 1969) or

TABLE 3 *P wave morphologies in paroxysmal supraventricular tachycardia patients* (see text)

Age range	18-29	30-39	40-49	50-59	60+	Total
No.	10	19	10	2	2	43
% -ve Ptf	60	47	30	100	50	49
% iso. Ptf	20	37	50	0	50	35
% +ve Ptf	20	16	20	0	0	16

mitral incompetence from papillary muscle dysfunction (Heikkilä, 1967). Our patients, however, all suffered relatively mild left ventricular disorder and had no clinical or x-ray evidence of cardiac failure. Seventeen per cent had a P wave terminal force more negative than -0.020 mm sec and 7 per cent had a force more negative than -0.030 mm sec. The majority of these cases had no other electrocardiographic abnormality at rest. The original study of Morris *et al.* (1964) suggested that a terminal force of -0.030 mm sec or less negative could be regarded as normal, and this limit of normality has been used in several further studies (Morris *et al.*, 1965; Gooch *et al.*, 1966; Saunders *et al.*, 1967; Heikkilä and Luomanmäki, 1970). However, we believe that -0.020 mm sec is a more acceptable limit, since only 1 out of 206 (½%) normal subjects had a P wave terminal force more negative, but 28 out of 159 (17%) of our ischaemic heart disease group had a terminal force more negative. It is interesting to note

FIG. 3 *Variation of the average P wave terminal force (Ptf) with age in the three diagnostic groups: paroxysmal supraventricular tachycardia, normal, and ischaemic heart disease.*TABLE 2 *P wave morphologies in ischaemic heart disease patients* (see text)

Age range	30-39	40-49	50-59	60+	Total
No.	7	33	64	55	159
% -ve Ptf	71	67	70	67	69
% iso. Ptf	29	30	20	27	25
% +ve Ptf	0	3	10	6	6

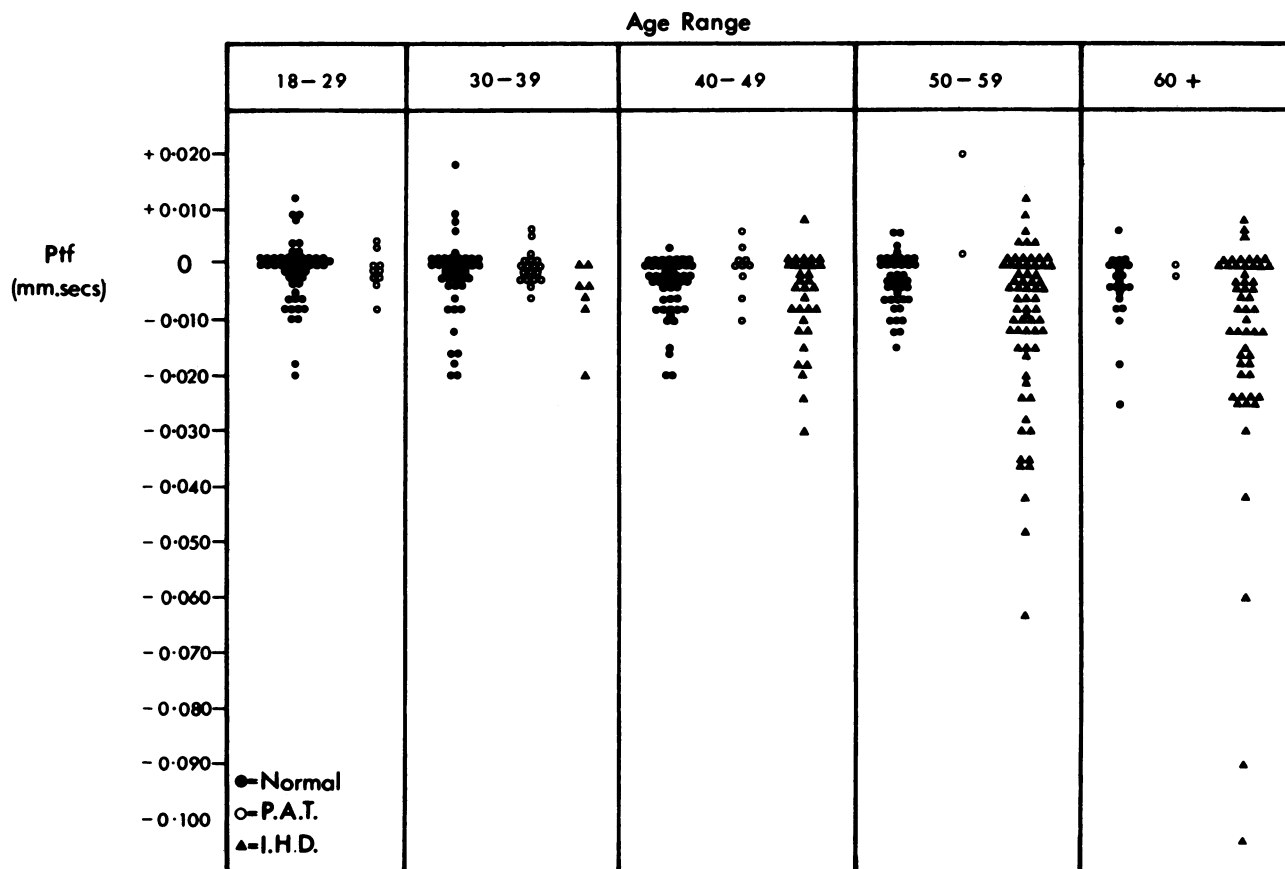


FIG. 4 A plot of the P wave terminal force (Ptf) for each subject in the study, indicating diagnosis and age range.

that our suggested normal value for healthy adults is similar to that which Reynolds (1967) found for healthy children. We think it likely that Morris *et al.* included some patients with occult ischaemic heart disease, whom we have taken as much care as possible to exclude, by careful history taking (Nixon and Bethell, 1971), examination in the left lateral position for the atrial gallop, and frequently using apex cardiography and phonocardiography and exercise testing.

Our study did not show an abnormal P wave terminal force in paroxysmal supraventricular tachycardia unless the patient suffered ischaemic heart disease as well. Davies and Ross (1963) found P waves of increased width or abnormal notching in 22 per cent of their series of patients with paroxysmal supraventricular tachycardia, with a higher incidence of these abnormalities in older patients. We had very few patients with simple paroxysmal supraventricular tachycardia over the age of

50; and it is also possible that Davies and Ross included some cases of occult ischaemic heart disease in their series.

The abnormal left atrial depolarization which caused the mitralization of the P waves in our cases of early ischaemic heart disease may be attributed to a number of factors. In general, left atrial enlargement correlates best with the size of an abnormal P wave terminal force (Sutnick and Soloff, 1962; Reynolds, 1967), but in each of our cases the left atrial shadow on a penetrated teloradiogram of the chest appeared normal. A second factor is hypertrophy of the left atrial muscle. In early ischaemic heart disease good evidence of increased left atrial activity is often found in the palpable and audible atrial gallop which results from augmentation of the atrial beat (Benchimol and Dimond, 1962; Banks and Shugoll, 1967; Ginn *et al.*, 1967; Nixon and Bethell, 1972). We have noted that the enlargement of the atrial component of the apex

displacement curve is accompanied usually by diminution of the rapid filling wave, and postulate that the primary haemodynamic abnormality of early ischaemic heart disease is an impairment of left ventricular filling in early diastole, probably from disease of the ventricular wall and/or increased end-systolic residual volume. This leaves a larger than normal volume of blood to be moved into the ventricle by atrial systole. This deficiency of early diastolic left ventricular filling, compensated by enhanced atrial activity, is probably similar to the situation in aortic stenosis where the size of the P wave terminal force (Sutnick and Soloff, 1963; Morris *et al.*, 1964) and the atrial gallop (Goldblatt, Aygen, and Braunwald, 1962) correlate well with the gradient across the aortic valve.

A third factor which may contribute to P wave abnormalities in ischaemic heart disease is degenerative fibrosis of the atrial wall from the coronary disease process (Pirani and Langendorf, 1949; Liu, Greenspan, and Piccirillo, 1961). Such a factor might explain the presence of abnormal P waves in cases where there is neither radiological evidence of left atrial enlargement nor mechanical evidence of an augmented left atrial beat.

It is interesting to note that mechanical evidence of pathological left atrial activity is nearly four times more common than electrical disorder of left atrial function in early ischaemic heart disease.

Conclusion

We conclude that a P wave terminal force more negative than -0.020 mm sec is a rare finding in normal subjects. This finding in a subject with no clinically detectable heart disease and an otherwise normal resting electrocardiogram should raise the suspicion of left ventricular dysfunction, of which ischaemic heart disease is the commonest cause.

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